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Foreign Animal Disease Report

USDA
United States
Department of Agriculture

Animal and Plant
Health Inspection Service

Veterinary Services

Emergency
Programs

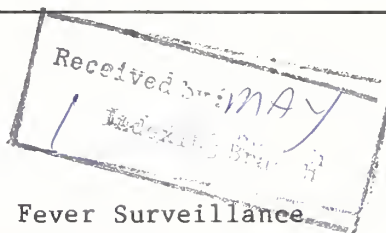


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In This Issue

Emergency Field Operations
Foreign Animal Disease Update
Caribbean Tick Program
Hog Cholera and African Swine Fever Surveillance
Mexico Foot-and-Mouth Disease Surveillance
Vesicular Stomatitis Virus Fingerprinting
Heartwater Update
Trypanosomiasis in the Western Hemisphere



Current Events

745
Emergency Field
Operations

During the period from October 1 through December 31, 1987, 50 emergency investigations of suspected exotic diseases were conducted by Veterinary Services (VS) veterinarians. No exotic diseases were found in 31 investigations for suspected vesicular disease, 7 for suspected African swine fever or hog cholera (HC), suspected avian influenza, exotic Newcastle disease or other avian diseases, and 4 investigations for suspected but unspecified exotic diseases.

An investigation was conducted as a result of the disclosure of a serum neutralization (SN) titer of 1:1024 for HC in a swine serum sample collected at slaughter at an abattoir in Texas. The serum came from a herd in which HC vaccine had apparently been used approximately 6 months previously. The serum was submitted as part of a disease surveillance program which involves the routine collection and testing of serum from swine slaughtered at abattoirs located in the Rio Grande Valley. Tonsil biopsies and tissue specimens were also submitted from seven swine in the herd. Six of the seven swine sampled reportedly had been vaccinated. All specimens were negative to fluorescent antibody tests of tissue sections and cell cultures. Serums from 24 pigs in the herd were collected and tested for HC and bovine viral diarrhea using the serum neutralization test. Only the serums from vaccinated pigs reacted to these tests.

Serums from 24 pigs in a neighboring herd were also negative to tests for HC. This herd had received a pig from the herd in which vaccine was reported to have been used.

Based on epidemiological evaluation, observation, history, and results of laboratory tests, HC was not present and had not occurred in the herds. The vaccinated animals, and those in contact with them, are restricted to sale and consumption in Texas.

In December, Texas reported that a lousefly from a cheetah in Tyler, Texas, had been identified as Hippobosca longipennis, at the Texas A&M College of Veterinary Medicine. The ranch on which the cheetahs originated had apparently received a male and four female cheetahs from Africa in 1985. Efforts are in progress to assist in the eradication of this fly. H. longipennis was confirmed twice before in the United States and was eliminated.

(Avian Influenza (AI) survey. During the period from March 1 through April 30, 1987, the U.S. Department of Agriculture, Animal and Plant Health Inspection Service, VS, conducted an extensive survey and testing program for AI in live bird markets and auctions in the United States. A total of 3,768 lots of cloacal swabs and 8,576 serum samples were collected from various species of birds. In tests at the National Veterinary Services Laboratories, Ames, Iowa, AI type H₅N₂ virus was isolated from three swab samples. Two were from chickens from Florida and New Jersey, and one was from a New Jersey pheasant. The viruses were not pathogenic in laboratory chickens. Other nonpathogenic serotypes isolated included H₁N₂ from a chicken and H₆N₈ and H₃N₈ from ducks.

Training and Assignments. During the period from November 16 to 20, 1987, 18 veterinarians from colleges of veterinary medicine and State diagnostic laboratories received training in foreign animal diseases (FAD) at the VS Foreign Animal Disease Diagnostic Laboratory, Plum Island, New York. Training planned for 1988 includes the following: military support course for military veterinarians, April 5-8; FAD seminar for FAD diagnosticians from the VS Central and Western Regions, May 24-26, 1988; Regional Emergency Animal Disease Eradication Organization (READEO) training for directors and field operations officers, June 14-16, 1988; Recorded Emergency Animal Disease Information (READI) system training, April 18-22, 1988; Wildlife Diseases course for FAD diagnosticians, September 6-9, 1988; and FAD course for diagnosticians, September 12-30, 1988.

In November 1987, two FAD diagnosticians received on-site ✓ experience in an outbreak of African horse sickness (AHS) in Spain. They reported that on September 12, 1987, AHS was confirmed in the provinces of Madrid, Toledo, and Avila in central Spain. In the eight localities affected by the disease, a total of 162 horses, mules, and wild asses died or were sacrificed. The origin of the outbreak was thought to have been from the importation of zebras from Namibia to a safari park located in the area of Aldea del Fresno, about 55 kilometers southwest of Madrid. A total of 37,000 units of modified live virus AHS vaccine of South African origin was administered to equines within the affected provinces. At the same time, an intensive effort was made to fumigate affected ranches and private and military stables and to control all movement of horses throughout the Spanish territory. The last horse death associated with the AHS episode reportedly occurred on September 28, 1987. Spanish animal health authorities felt that the epidemic was under control by the end of September and that unrestricted movement of horses would be reinstated by early

Foreign Animal
Disease Update

November. However, deaths of vaccinated horses in the Madrid area that occurred at the time of this report (end of October 1987) still await a definitive diagnosis. Subsequently, a report to the Office International des Epizooties October 29, 1987, indicated that no new cases had been found since October 23, 1987. At that time a race horse was sacrificed in the Madrid area after it exhibited symptoms and was diagnosed as being infected with AHS. This occurred 34 days after the horse was vaccinated against the disease. (Dr. M. A. Mixson, 301-436-8073)

In Europe, an outbreak of foot-and mouth disease (FMD) type O₁ was reported in the Riga District of the Latvian S.S.R. in November 1987. Six of 99 bovines had mouth lesions. The most recent prior outbreak of FMD in Latvian S.S.R. reportedly was in November 1986. Soviet authorities reported that all 99 bovines exposed to the disease were destroyed. The source of the outbreak may be related to a nearby game farm where wild animals were fed raw by-products from vaccinated animals.

The world reference laboratory in Pirbright, England, reported the following occurrences of FMD July through October 1987: Type O in Bangladesh, Egypt, Laos, Sri Lanka, and Yemen; Type A in Saudi Arabia and Turkey; Type SAT₁ in Zambia; Type SAT₂ in Zimbabwe; and Type Asia₁ in Bangladesh.

In South America, FMD virus types O₁, A₂₄(S. Carlos), A₈₁(Argentina 87), and C₃ were identified in Brazil, and types O₁, and A₂₄ in Colombia. In Chile, FMD was last reported in August 1987. A total of 81 outbreaks was reported in that country, and 31,386 animals were destroyed.

During July, August, and September 1987, 252 herds were reportedly affected with vesicular disease in Brazil, 149 in Colombia, 9 in Ecuador, 46 in Uruguay, 33 in Venezuela, and 8 in Chile.

In Southern Africa, Botswana reported an outbreak of African swine fever (ASF) during late November 1981, in which 54 of 315 pigs died. The remaining swine on the premises were depopulated. Argasid ticks (Ornithodoros moubata porcinus) were thought to have transmitted the virus from wild warthogs to domestic pigs. Spain, Italy, and Portugal continue to report cases of ASF.

Sri Lanka reported an outbreak of suspected Rinderpest (RP) in which 400 cattle were affected and 3,000 exposed. The first clinical evidence of the disease occurred October 24, 1987. Goats imported into the country from India during September 1987 are suspected as the source of the disease. The most recent prior report of RP in Sri Lanka was in 1942. The Western African country of Ghana reported several outbreaks of RP during September and October 1987, in which 469 of a total of 4,692 affected cattle died. These outbreaks were thought to be related to the illegal importation of cattle from adjacent, infected countries.

In Western Africa, Burkina Faso (formerly Upper Volta) reported an outbreak of **contagious bovine pleuropneumonia** (CBP) during September 1987. A herd of 115 animals was destroyed after CBP was diagnosed in 14 animals. (Dr. Percy Hawkes, 301-436-8285)

246
Caribbean Tick
Program 10

This article updates a report published in the Spring 1987 issue (15-1) concerning a feasibility proposal to manage the tropical bont tick (TBT), Amblyomma variegatum, and its associated diseases--heartwater and acute dermatophilosis--in the Caribbean. The completed proposal was sent through the Inter-American Institute for Cooperation on Agriculture (IICA) to interested organizations and governments on March 1, 1987. A technical workshop on the management of the tropical bont tick and associated diseases followed March 17-19 on the island of Barbados. It was attended by representatives from many Caribbean islands, the U.S. Department of Agriculture (USDA), the Agency for International Development (AID), IICA, the Food and Agriculture Organization of the United Nations (FAO), and other public and private organizations. Resolutions were approved to: (1) establish an Amblyomma program council under the purview of CARICOM (Economic Community of the Caribbean Countries), (2) establish a pilot project on an island to demonstrate tick eradication techniques, (3) convene a conference to present the program to potential donors, and (4) support current tick control activities by FAO and other organizations.

In fiscal year (FY) 1987, Congress authorized an expenditure of no less than \$2 million by AID for the control and eradication of TBT in the Caribbean. AID and USDA agreed to use this money and other resources to carry out a demonstration eradication project on Antigua.

In July, a team of scientists put together by the consortium for International Crop Protection carried out an environmental assessment of the possible effects of using acaricides in a tick eradication program on Antigua. On September 30, USDA and AID signed an agreement for the demonstration project on Antigua. The project has three central components: (1) an eradication component to be carried out by the USDA's Animal and Plant Health Inspection Service (APHIS), (2) information and evaluation components consisting of a wildlife study, an economic impact analysis, and an outside evaluation to be implemented by the USDA's Office of International Cooperation and Development (OICD) through several collaborators, and (3) a policy and strategy formulation component to be conducted by AID. OICD also plans to support cooperative studies of tick biology and control on Guadeloupe by USDA and French scientists.

APHIS is now developing plans and agreements with the Government of Antigua for a TBT Eradication Demonstration Program. The eradication plan has four phases: (1) planning, (2) preoperations (training, surveillance, facility construction, and procurement of vehicles and acaricides), (3) eradication operations of applying acaricide to large animals biweekly, and (4) surveillance to determine if all ticks have been eliminated from the island. Acaricides selected are Amitraz for cattle and Permethrin for horses, cattle, and sheep.

The Standing Committee of the Ministers of Agriculture of the CARICOM member countries met in Rome during November 1987 to establish an Amblyomma Steering Committee. The committee will provide a forum for the encouragement and promotion of Amblyomma eradication in the Caribbean. (R. O. Drummond, Kerrville, Texas, 512-257-3566; J. O. Butcher, USDA-OICD, Washington, D.C., 202-653-7462)

246
Hog Cholera/
African Swine
Surveillance //

The following article updates information reported by Dr. G. A. Erickson in the Fall 1987 issue (15-3:3-4).

Although the United States and the Commonwealth of Puerto Rico are free of hog cholera (HC), the disease remains endemic in the swine population of much of the rest of the world. In the Western Hemisphere, only Canada and the United States are free of HC. The risk of introduction is, therefore, very great and justifies the surveillance system that is currently in use. Greatest risk for introduction into the Continental United States is incurred by the illegal introduction of infected swine into Texas from Mexico. Despite the manufacture and sale of many types of vaccine, HC is a major health problem for Mexican swine. Clinical cases frequently occur in the Mexican States bordering the United States. The other important risk for the introduction of HC is the feeding of commercial garbage to swine, a practice most prevalent along the eastern seaboard of our country. Feeding uncooked table scraps of pork or illegally imported fresh or poorly cured pork products to garbage-fed swine is considered the second most likely means of introduction of HC.

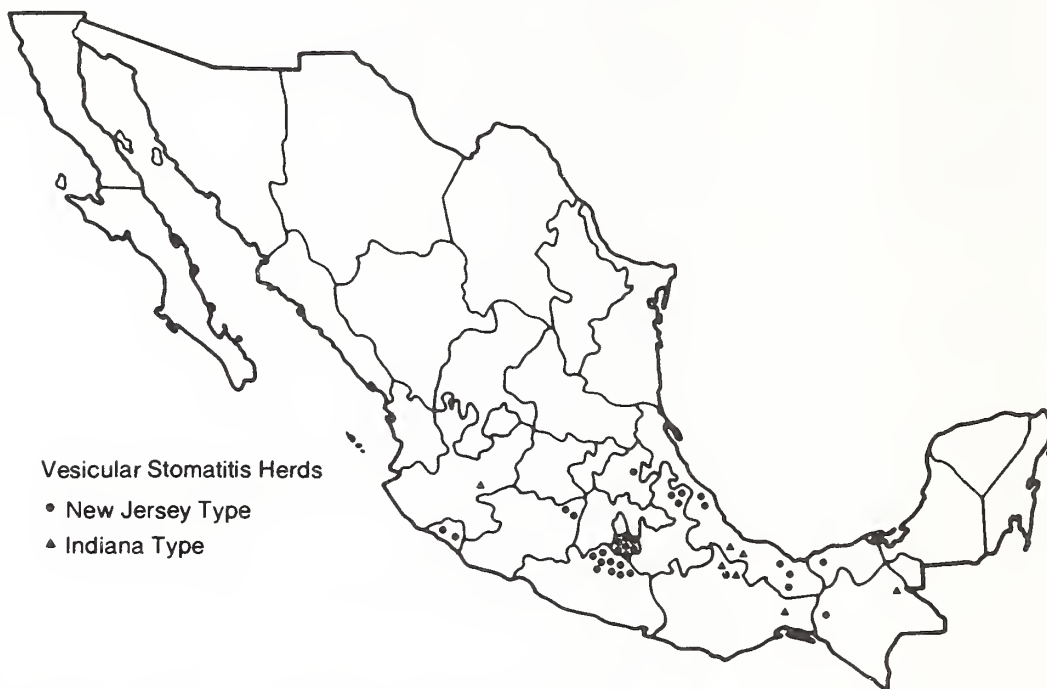
Accordingly, the USDA's HC surveillance program is focused on Texas; the major garbage-feeding States of New Jersey, Massachusetts, Rhode Island, and New Hampshire; and the Commonwealth of Puerto Rico.

Sera from slaughter swine raised along the Rio Grande River and around the major cities of Philadelphia, New York, and Boston are randomly sampled at the rate of 500 to 700 sera per month, or 6,000 to 10,000 sera per year, and are submitted to the National Veterinary Services Laboratories (NVSL) for HC serological testing. Puerto Rico submitted over 16,000 sera during the period from October 1, 1986-September 30, 1987. More than 6,000 garbage feeders' premises in the United States and Puerto Rico are regularly inspected for compliance with Federal licensing requirements and for swine health. (Dr. G. A. Erickson, NVSL, USDA, Ames, Iowa, 515-239-8551)

246
Mexico Foot-and-
Mouth Disease
Surveillance //

Mexico continues to be free of foot-and-mouth disease (FMD), according to the Mexico-United States Commission for the Prevention of Foot-and-Mouth Disease and Other Exotic Diseases (CPA). Surveillance activities by the CPA included the investigation of 107 reports of vesicular disease in 1986 and 190 in 1987. New Jersey type vesicular stomatitis (NJVS) was found on 34 premises in 1986 and on 39 in 1987. Seven were affected with Indiana type vesicular stomatitis (INVS) in 1986 and 31 in 1987. The following maps show the distribution of VS in 1986 and 1987: (See reports of prior years in 14-1, 1986, and 13-1, 1985.)

Mexico—1986



Mexico—January 1-December 21, 1987



Vesicular stomatitis exhibits a cyclical behavior in Mexico. The disease is seen every year in the same general area, recurring within this area at different locations on 3- to 5- and up to 10-year cycles.

In 1986, NJVS was seen in the States of Guerrero, Morelos, Puebla, and Veracruz, with scattered, sporadic cases in Colima, Michoacan, Oaxaca, Hidalgo, Chiapas, and Tabasco. There were small outbreaks of INVS in Oaxaca and Veracruz and scattered cases in Jalisco and Chiapas. The 1986 activity included a sudden outbreak of NJVS in Guerrero from August to November and two isolated outbreaks after that, one in Tabasco at the end of November and another in Michoacan at the end of December. This was followed by a hiatus until the end of March 1987, when two premises were affected in Guerrero. Sudden outbreaks of NJVS were then seen in Chiapas in June, July, August, and September; in Tabasco in September, October, November, and December; and in Campeche in November 1987.

The last outbreak of INVS in 1986 was seen in Veracruz in September. In 1987, sudden outbreaks were seen in Puebla, Oaxaca, and Veracruz in January, February, March, and April; in Chiapas in May, September, and November; and in Nayarit in December. During 1987, both NJVS and INVS were found in cattle concurrently on two separate premises.

Cattle were principally affected with VS in both 1986 and 1987. Horses were affected with NJVS on six premises in 1987, and with INVS on one premise in 1986 and on five premises in 1987. Swine were affected with NJVS on one farm in 1986 and on two farms in 1987.

In bovines with NJVS, vesicular lesions were seen mainly in the mouth (70 percent of the herds). Lesions were seen only on the teats in 10 percent of the herds. With INVS, lesions were seen only in the mouth in 55 percent of the affected herds and only on the teats in 40 percent of the herds. Cattle in two herds with INVS were reported to have generalized lesions (mouth, teats, and feet).

In the bovine herds affected with NJVS, the attack rate (percent of animals with lesions) was 7.8 percent in 1986 and 12.0 percent in 1987, as compared to 8.0 percent in 1985 and 6.1 percent in 1984. For INVS-affected herds, the attack rate was 12.4 percent in 1986 and 5.4 percent in 1987. Actual attack rates were probably somewhat higher, since not all the mouths of animals were examined, and some may have had lesions without showing any excess salivation or frothing.

In 1986, one bovine was reported to have died in a herd during a NJVS outbreak and in 1987, one died in an INVS-affected herd. In 1987, 12 of 23 bovines affected with INVS reportedly died. Most of these were calves. The possible role of VS in causing these deaths was not determined.

In 1986, 9 of the 34 premises with NJVS reported having vesicular

disease in previous years. None of the premises with INVS reported previous cases. In 1987, 8 of the 39 premises with NJVS reported having cases of vesicular disease previously, and 10 of the 31 premises with Indiana VS reported previous cases. In 1986, 30 percent of the owners of herds with VS reported other herds affected in the vicinity, as compared to 65 percent in 1987.

In 1986, two herds affected with NJVS had added new animals within the month before the onset of VS. In 1987, three herds with NJVS and three with INVS had added new animals before the outbreak. A probable source of the VS outbreaks could not be determined.

In 1987, the average period between the onset of a VS outbreak in a herd of bovines and its investigations was 7.2 days. An additional 2 days were needed to send specimens to the CPA Vesicular Disease Laboratory and have the necessary tests made. Efforts are being made to reduce this time period to help prevent the spread of possible FMD before eradication measures can be taken. (Dr. John Mason, AG/AFT, Mexico City, U.S. Department of State, Washington, DC 20520)

246.
Vesicular
Stomatitis
Virus

Fingerprinting

Historically, epizootic waves of vesicular stomatitis have been seen in the United States at about 10-year intervals. (See December 1983 issue, 10-3:8-14.) However, a different pattern has appeared in recent years. Vesicular stomatitis virus (VSV) of the New Jersey (NJ) serotype, first isolated in 1949, has been identified in the United States annually for the last 5 years. A major epizootic affecting cattle and horses swept through the western States in 1982 and 1983, causing considerable financial losses and trade restrictions. At that time, virus isolations and wild swine seroconversions were being reported on Ossabaw Island, off the coast of Georgia. In 1984, VSV was isolated from cattle in Texas. Unlike the historical pattern, another epizootic affecting cattle and horses erupted in Colorado, New Mexico, and Arizona in 1985. VSV NJ was again isolated in Colorado from cattle and horses in 1986, and on Ossabaw Island, virus isolations and seroconversions were reported in wild swine in 1987.

To begin to try to answer some important questions raised by the variable epidemiological characteristics of VS that have been observed in recent years, researchers at the University of Nevada initiated the molecular analysis of VSV evolution and VSV epizootiology. These studies have been supported through competitive research grants on biotechnology and animal molecular biology from the U.S. Department of Agriculture (USDA) and the collaborative efforts of Drs. E. A. Carbrej and G. A. Erickson at National Veterinary Services Laboratories, Ames, Iowa, and Dr. J. Mason and colleagues at the Mexico-U.S. Commission for the Prevention of Foot-and-Mouth Disease, Mexico City, Mexico. Over the past 3 years, the Nevada researchers have analyzed over 100 NJVS viruses that were isolated in the United States, Mexico, and Central America, using the T1 ribonuclease fingerprinting technique (S.T. Nichol, 1987. J. Virol., 61:1029-1036, and 1988

Summarized data indicate the existence of at least 14 distinct genotypes of VSV NJ. The following map shows the geographical location of T1 fingerprint groups 1-10, isolated during the period from 1982 to 1987:



Most of the isolates from the United States and Mexico (over 50 isolates) constituted a single T1 fingerprint group (group 1). Comparison of fingerprints within this group led to the following conclusions: (1) Viruses within each successive western United States outbreak (1982-83, 1984, and 1985-86) were relatively homogeneous genetically. This was apparent even when the viruses compared had been collected at different stages of an outbreak, from different States, from various hosts including cattle,

horses, swine, and insects, and from different host lesion sites. (2) Virus activity in 1982 and 1983 was part of an ongoing outbreak which had continued throughout the winter months. (3) Viruses isolated in 1984 from Texas showed numerous differences from the 1982-83 viruses and represented a separate outbreak. (4) Viruses isolated in 1985 and 1986 from Colorado, New Mexico, and Arizona showed numerous differences from those isolated during 1982-83 in the western United States, indicating a separate origin. (5) Viruses obtained from Colorado in 1986 had T1 patterns identical to those of many of the 1985 isolates, strongly suggesting that the virus had overwintered in the southwestern United States, i.e. the 1986 virus activity was a continuation of the previous year's activity. How the virus was maintained during the winter months is unknown. No signs of vesicular diseases in livestock were reported during the winter of 1985-86.

Analysis of VSV NJ viruses from Mexico, isolated either prior to or concurrent with outbreaks in the United States, revealed fingerprint patterns that were very similar and, in some cases, identical to their U.S. counterparts. These findings, together with the results of epidemiological investigations indicating a northward spread of virus activity during the early stages of the 1982-83 VSV NJ outbreak, and the existence of enzootic zones of VSV NJ in Mexico, strongly suggest that Mexico may be the source of the VSV NJ found in recent disease outbreaks in the United States.

Ossabaw Island, off the coast of Georgia, is the only other well documented enzootic zone of VSV NJ in North America. (See 11-4:1-2.) Dr. V. Nettles and colleagues at the University of Georgia and Dr. G. A. Erickson at NVSL have carried out extensive serological and virus isolation studies in this area during the past 5 years. The annual seroconversion of wild and sentinel swine and the isolation of NSV NJ in 1983 and 1987 demonstrated the enzootic nature of the disease in this region. T1 fingerprint analysis of these viruses indicated they were unrelated to those from the western United States or Mexico. Three 1987 Ossabaw isolates were found to have identical T1 fingerprints and were different from the 1983 Ossabaw isolate at only four T1 spots. VSV NJ of T1 group 2 is apparently being maintained in a relatively stable manner on the island.

In addition to the T1 group 1, two other T1 fingerprint groups have been identified from Mexico. Virus groups 3 and 4 were identified from Veracruz and Oaxaca, respectively. The regions in which the viruses were collected are considered to be enzootic disease zones. The presence of three virus groups (1, 3, and 4) in this relatively small region, as compared to only one group in the vast epizootic regions of the United States and Mexico, suggests that a higher degree of genetic diversity of VSV NJ exists in the more southerly enzootic disease zones. This became even more evident on analysis of Central American VSV NJ isolates.

VSV NJ isolates from Central America have been serologically

characterized by the Laboratory for the Diagnosis of Vesicular Disease (LADIVES) in Panama, headed by Dr. L. Roquebert. Dr. R. B. Tesh, Yale Arbovirus Research Unit (YARU), provided the Nevada laboratory with these viruses for comparison. Six distinct T1 fingerprint groups of viruses (groups 5 through 10) were found to exist in Central America from 1982 to 1985. This is a rather remarkable genetic diversity for the relatively small enzootic disease zone involved. Virus groups 5 through 10 are clearly distinct from those identified in the United States and Mexico, indicating that the disease process in this region is unrelated to that occurring further north.

Four older VSV NJ groups have been identified, each consisting of a single member: the Ogden strain, which was originally isolated from a cow in Utah in 1949; the Hazelhurst strain, originally isolated from a pig in Georgia in 1952; a 1960 strain isolated from a bovine in Panama; and a 1976 strain isolated from mosquitoes in Ecuador. It is becoming clear that the VSV NJ serotype should not be thought of as a homogeneous entity but should be considered a collection of serologically related but genetically variant viruses.

The University of Nevada possesses a large data base of T1 fingerprint catalogs for VSV NJ isolates, making it possible to ascertain the interrelationships of various vesicular stomatitis outbreaks throughout North and Central America. The genetic differences among VSV NJ serotype viruses provide a means of identifying transmission routes and virus reservoirs, and are currently being exploited to provide a basis for the rapid and precise differential diagnosis of these viruses, using a panel of synthetic DNA probes. (Dr. S. T. Nichol, Cell and Molecular Biology Program, University of Nevada, Reno, NV 89557, 702-784-4250) ✓

2405
Heartwater
Update //

Heartwater, an acute noncontagious tick-borne disease of domestic and wild ruminants caused by Cowdria ruminantium, was reviewed in the June 1982 issue (10-1). The diagnosis of heartwater on the islands of Guadeloupe, Marie Galante, and Antigua and the distribution of the vector, Amblyomma variegatum, in the Caribbean, were discussed in the March 1985 issue (13-1). Subsequent to these reports, considerable additional knowledge has been obtained from the studies of Dr. Linda Logan at the Plum Island Animal Disease Center and from studies by others who participated in a meeting entitled "Heartwater, Past, Present, and Future," in South Africa during October 1986. Highlights of the recent research are reviewed in this article. ✓

Etiologic agent. There are many isolates of C. ruminantium. Some isolates e.g. Kumm, Kwanyanga, and Nonile replicated in both ruminants and the mouse. Others, e.g. Ball 3, Gardel, and Mali, replicated only in ruminants. In cross protection studies, serological difference were found between the isolates. The greatest difference was between those isolates that replicated in the mouse and those that did not. C. ruminantium was also found to have a strong serological relationship to the granulocytic Ehrlichia equi and, to a lesser degree, to E. canis.

South African workers have been able to propagate C. ruminantium in an endothelial cell culture.

Vectors. Heartwater is spread by ticks of the Amblyomma spp. In Africa, 10 species of Amblyomma have been shown to be vectors. Species are not equally important in transmission. In the Western Hemisphere, ticks with a demonstrated capacity to transmit C. ruminantium are A. variegatum, A. maculatum, and A. cajennense. Even though heartwater in the Western Hemisphere has been reported only from three Caribbean islands, the potential for C. ruminantium to persist and spread exists also in areas ranging from the southern United States to the northern part of South America because of the presence of A. maculatum. A similar potential exists in areas ranging from southern Texas to South America because of the presence of A. cajennense.

Only the ticks that feed during the height of the febrile response become infected. The efficiency of transstadial transmission of C. ruminantium varies between species. South African workers recently demonstrated transovarial transmission in a very small number of A. hebraeum ticks.

Host susceptibility. Heartwater is usually thought of as a disease of domestic ruminants. However, C. ruminantium can replicate in many host species. In domestic ruminants, there are significant differences in susceptibility between breeds of the same species, e.g. in Guadeloupe the Kraal goat is more resistant than the Dutch breeds, and in South Africa, Angora goats are very susceptible, and Jersey and Brahma breeds are more susceptible than other breeds of cattle. Many of the native species of African antelope are susceptible but develop only a very mild or subclinical infection. Other native antelope are resistant. Buffalo, bison, and white-tailed deer are susceptible.

South African workers have shown that sufficient replication will occur in the tortoise and guinea fowl to infect Amblyomma feeding on these animals. Replication has also been demonstrated in the ostrich.

Infection of white-tailed deer and subsequent endemic infection of Amblyomma populations would make eradication from North America very difficult.

Diagnosis. The primary method of confirming a diagnosis of heartwater has been the demonstration of C. ruminantium organisms in the cytoplasm of capillaries in Giemsa stained brain smears made from a dead animal. This is still a recommended procedure. A tentative diagnosis in a live animal can be confirmed by demonstrating the organism in a Giemsa or immunofluorescent stained smear made from a needle biopsy of the cerebral cortex and in neutrophilic leukocytes cultured from the blood.

Dr. Logan found that neutrophilic leukocytes cultured from the blood of a febrile animal could be used as an antigen for an indirect immunofluorescent test. This was the antigen used to determine the relationship of C. ruminantium to other rickettsia

and was used to screen sera from ruminants on several Caribbean islands for antibody to C. ruminantium.

Treatment. If the disease is suspected when the only sign is fever, two doses of 5mg/kg of tetracycline given intramuscularly at a 24-hour interval or one dose of 20 mg/kg of long acting tetracycline given intramuscularly will suppress the disease while allowing immunity to develop. Once neurological signs develop, the prognosis is poor. (C. A. Mebus, USDA, ARS, Plum Island Animal Disease Center, 516-323-2500)

Focus on...

245 **Trypanosomiasis in the Western Hemisphere**

Trypanosomiasis has long been recognized in Africa as an economically devastating disease of domestic cattle and small ruminants. While several protozoans of the genus *Trypanosoma* are responsible, only one, *Trypanosoma vivax*, has spread to the Western Hemisphere. In the Western Hemisphere, the parasite is known to be a pathogen of cattle and water buffalo, causing both acute and chronic disease.

Geographic Distribution

In Africa, *T. vivax* is found mostly within the range of its biological vector, the tsetse fly (*Glossina* spp.). The parasite was first brought to the Western Hemisphere in the late 1800's, in a shipment of infected bulls from Senegal, Africa. *T. vivax* was then disseminated and became established throughout the South American continent without benefit of the tsetse. Transmission was probably mechanical, by biting flies, when infected and uninfected animals were mixed. The first official report of this parasite in South America was from French Guiana in 1919. Infected cattle from Venezuela and Colombia apparently carried the parasite to the islands of Guadeloupe and Martinique in the French West Indies during the late 1920's. *T. vivax* continued to spread throughout South America, and by the 1930's, it had spread into the Central American countries of Panama, Costa Rica, and El Salvador. Argentina is the only South American country that has not reported the occurrence of this parasite. The most recent report of an outbreak of *T. vivax* in the Western Hemisphere (1970-79) was in the neighboring municipalities of Manzanillo and Bayamo, Cuba, less than 100 miles off the shores of the Florida Keys. A recent serologic survey conducted by the University of Florida found Puerto Rico and 12 of 14 islands sampled in the Lesser Antilles free of *T. vivax*. Three animals on two islands were found to be "suspect," but the presence of the parasite has not been demonstrated conclusively at this time. The same study showed that no islands appear to have *T. vivax* within their sheep and goat populations.

Hosts

In Africa, *T. vivax* causes clinical disease in a variety of livestock, including cattle, sheep, and goats, with various species of antelope acting as asymptomatic carriers. However, in South America, only domestic cattle and water buffalo seem to be important hosts. The *Bos taurus* breeds, particularly those recently imported from Europe, appear to suffer most. An increased plane of nutrition may reduce the impact of infection

in these breeds. The Bos indicus (zebu) breeds and their crosses seem better able to resist the intermittent waves of parasitemia in chronic infection and apparently suffer less than the Bos taurus breeds from the acute disease. Sheep, goats, and horses can be experimentally infected, but there are no published reports of naturally occurring infections in these species in the Western Hemisphere. The existence of wild reservoir hosts in the Western Hemisphere is open to question.

Transmission

The primary means of transmission of pathogenic trypanosomes in African livestock is by the tsetse fly. This fly acts as a true biological vector, allowing development and amplification of the trypanosome within its salivary glands. When T. vivax spread to the Western Hemisphere, it left the tsetse behind and adapted to mechanical transmission. Tabanids and Stomoxys have long been hypothesized as the most likely mechanical vectors due to their nervous feeding behavior. The painful bites inflicted by these flies result in attempts by the animal to dislodge the flies, thus interrupting feeding. The parasite may then be transmitted via contaminated mouth parts as the fly re-initiates feeding on a different host. Previously, mechanical transmission had not been demonstrated conclusively in the Western Hemisphere, and T. vivax was not found during the dissection of over 1,000 tabanids collected while feeding on infected cattle in Colombia. However, work recently conducted in French Guiana by a joint effort of the University of Florida and INRA (French National Institute of Agricultural Research) has shown that the tabanid, Cryptotylus unicolor, does transmit T. vivax under experimental conditions and that the parasite can survive at least 5 hours in the midgut of both C. unicolor and Stomoxys calcitrans. Other dipteran species in the families Culicidae and Simuliidae have also been proposed as potential vectors. In Cuba, a list of vector species which transmit T. vivax has been published and includes, in addition to those mentioned above, the family Ceratopogonidae (gnats). The list omits Tabanidae. However, exactly how this list was developed by Cuban researchers is not clear.

Transmission of T. vivax in the Western Hemisphere appears to be relatively inefficient. It is likely that a combination of events is necessary for transmission and endemic maintenance of the parasite, i.e. (1) a high concentration of cattle in a small area, (2) large numbers of biting flies, and (3) at least a few cattle with a high parasitemia.

Pathogenesis

T. vivax infection may produce either acute or chronic disease in cattle. Acute disease occurs almost exclusively in adult cattle and is usually seen when the parasite invades a susceptible herd or when susceptible animals are brought into an endemic area. Clinical signs include severe anemia, fever, dramatic weight loss, edema, hemorrhage, and death. The reproductive system is frequently affected, and abortion, stillbirths, and infertility are common in endemic regions. Pregnant animals may abort or deliver stillbirths and cease lactation. Subclinical or chronic infection may occur also, producing emaciation, decreased milk production, cardiac insufficiencies, and muscle atrophy. Once

the parasite has established itself within a geographic area, it is very difficult to assess the pathogenesis and economic impact because chronically infected animals are generally considered "poor doers" and are likely to suffer concurrently from other diseases.

There is strong evidence for immunosuppression in animals chronically infected with T. vivax. A tenfold reduction in the ability of the host to respond to other antigens has been reported. This may reduce the success of vaccination campaigns (i.e. foot-and-mouth disease and rinderpest) and increase the severity of secondary or concurrent infections, such as anaplasmosis and babesiosis. Chronically infected animals also become reservoirs for the spread of the parasite. Periods of high stress such as dehydration, malnutrition, or pregnancy compromise host immunity and allow for rapid multiplication and dissemination of the organism.

Diagnosis

Diagnosis of T. vivax infection is very difficult, except during acute disease when a high parasitemia is present. Clinical signs are nonspecific and mimic other common diseases of cattle in the tropics such as babesiosis and anaplasmosis. Frequently, all three organisms are found in a single animal. During the natural course of both acute and chronic infections, parasitemia recurs in waves. Trypanosome numbers rise with the appearance of each new antigenic variant and fall as the animal mounts an immune response to that variant. The duration of periods of high parasitemia is variable. In recent experimental studies in French Guiana, detectable parasitemias lasted from 36 hours to 7 days. When parasitemia is at a peak, diagnosis by thick or thin blood smear is possible, but when the parasitemia declines, direct parasitological diagnosis becomes very difficult. Several techniques have been developed which effectively concentrate the organisms to facilitate the diagnosis of a low level parasitemia. Subinoculation into laboratory animals, anion-exchange chromatography, and microhematocrit centrifugation are successful techniques for several species of *Trypanosoma*. Unfortunately, subinoculation and anion-exchange chromatography have generally proven unsuccessful for detection of T. vivax. The fastest, simplest, and most sensitive method for diagnosis of both acute and chronic T. vivax infections is the microhematocrit centrifugation technique of Murray (1977, Trans. Royal Soc. Trop. Med. Hyg., 71:325-326). With this technique, it is possible to detect a parasitemia as low as 10^2 to 10^3 trypanosomes/ml. This technique is currently in use in French Guiana for the diagnosis of both acute and chronic T. vivax infections in individual animals and herds.

A potential complicating factor in parasitological diagnosis is the existence in South and Central America of another mechanically transmitted trypanosome of livestock, T. evansi. This species causes an often fatal disease in equids in South America and camels in the Eastern Hemisphere. Cattle can serve as asymptomatic carriers. The two species of trypanosomes are nearly identical when viewed by light microscopy. The single distinguishing difference lies in the movement of the parasites

in a fresh wet blood film. T. vivax exhibits a rapid, anteriorly directed movement, coming into a field of view and then disappearing within a few seconds, its path often a straight line. Trypanosoma evansi merely undulates rapidly in place.

Serological techniques have been developed to diagnose chronic infections and define endemic areas. The indirect fluorescent antibody (IFA) test and enzyme-linked immunosorbent assay (ELISA) are currently the methods of choice. Serological diagnosis is not definitive, however, and difficulties in interpretation arise because cross reactions with other pathogenic trypanosomes (especially T. evansi, in the Western Hemisphere) are common. Successfully treated animals may remain serologically positive for some time after chemotherapy. Both parasitological and serological testing are necessary to confirm the diagnosis of trypanosomiasis in an individual or herd.

Treatment and Control

Several compounds are effective in the treatment of acute or chronic trypanosomiasis, but prophylaxis has proven difficult. Diminazene aceturate (Berenil), the chemotherapeutic used most commonly, is a non-toxic, easily obtained and easily administered, water soluble compound. It is given intramuscularly at a curative dose rate of 3.5 mg/kg. It is not licensed for general use in the United States. Resistance of various Trypanosoma species to Berenil is known to occur in Africa, but has not yet been documented in South America. Unfortunately, curative treatment renders the animal or herd completely susceptible to reinfection and acute disease. Serologic studies indicate that antibody titers (and presumably protection) persist for, at most, several months after treatment before declining to preinfection levels. If herd treatment is to be instituted, strict control measures must be implemented to prevent reintroduction of the parasite.

Summary

Little is actually known about the epizootology of T. vivax in the Western Hemisphere. Transmission is thought to occur mechanically by ubiquitous biting flies, primarily tabanids and Stomoxys, but this has not been proven conclusively. Introduction of the parasite into a herd of susceptible adult cattle or the movement of cattle into an endemic area can result in abortion, stillbirth, cessation of lactation, weight loss, and up to 40 percent mortality within several weeks. The economic impact of the disease in an endemic area is difficult to assess and has not been estimated.

In view of the lack of information about T. vivax in the Western Hemisphere and reports of its presence as close to our shores as Cuba, consideration must be given to the potential for the introduction and spread of this parasite into and within the Continental United States. (Dr. Susan A. Ferenc, University of Florida, Gainesville, FL 32610-0137; 904-392-1841)

Questions about the FAD Report may be sent to:

Dr. Edwin I. Pilchard, Editor
USDA, APHIS, VS
Room 801, Federal Building
6505 Belcrest Road
Hyattsville, MD 20872

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